(FILE 'HOME' ENTERED AT 17:25:16 ON 15 SEP 1999)

FILE 'CAPLUS' ENTERED AT 17:29:15 ON 15 SEP 1999

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L1          260 S ((FORMYL(W)METHION?) OR FORMYLMETHION?) (2W) PEPTIDE
L2            2 S L1 AND ALLERG?
L3            0 S L1 AND ASTHMA
L4         2059 S ALLERG? AND MAST CELL
L5          397 S ALLERG? AND (MAST CELL) AND REVIEW/DT
L6          124 S L5 AND CYTOKINE
L7          122 S L6 NOT VACCINE?
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=> d bib, abs 20-40

L7 ANSWER 20 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1998:372757 CAPLUS
DN 129:53129
TI Recent research for the development of antiallergic agents. An approach for the regulation of **allergic** functional molecule
AU Nagai, Hiroichi
CS Dep. Pharmacol., Gifu Pharm. Univ., Gifu, 502, Japan
SO Arerugi (1998), 47(5), 475-483
CODEN: ARERAM; ISSN: 0021-4884
PB Nippon Arerugi Gakkai
DT Journal; **General Review**
LA Japanese
AB A review with 21 refs., on regulation of **allergic** functional mols., e.g., **cytokines**, IgE, arachidonate, and histamine, which are target mols. of anti-**allergic** drugs. Receptor-mediated signal transduction pathway of **allergic** functional mols., and activation mechanism of the **allergic** cells are also discussed.

L7 ANSWER 21 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1998:331204 CAPLUS
DN 129:80269
TI Molecular basis of **allergic** diseases
AU Leung, Donald Y. M.
CS Division of Pediatric Allergy and Immunology, The National Jewish Medical and Research Center, Denver, CO, 80206, USA
SO Mol. Genet. Metab. (1998), 63(3), 157-167
CODEN: MGMEFF; ISSN: 1096-7192
PB Academic Press
DT Journal; **General Review**
LA English
AB This review with 91 refs. examines the current understanding of the mechanisms underlying **allergic** diseases. The IgE mol. plays a central role in the pathogenesis of immediate hypersensitivity reactions by virtue of its capacity to bind specifically to high-affinity IgE

receptors on **mast cells** and mediate the release of various **mast cell**-derived mediators and proinflammatory **cytokines** on exposure to **allergen**. Clin. significant **allergic** responses are followed by a late-phase response dominated by eosinophils and T lymphocytes. The majority of T cells in **allergic** responses are memory T cells secreting helper type 2 (TH2)-like **cytokines**, i.e., interleukin (IL)-4, IL-5, IL-13, but not interferon-.gamma.. These **cytokines** regulate IgE synthesis and promote eosinophil development, thus contributing to **allergic** inflammatory responses. Failure to control immune activation early in the course of **allergic** disease blunts responses to glucocorticoid therapy and contributes to disease progression. The identification of key cells and mols. involved in the initiation and maintenance of **allergic** inflammation is likely to become an important target in the treatment of this common group of illnesses.

L7 ANSWER 22 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1998:224838 CAPLUS
DN 128:293690
TI The TH1/TH2 paradigm in **allergy**
AU Maggi, Enrico
CS Clinical Immunology Dept., Istituto di Medicina Interna e Immunoallergologia, University of Firenze, Florence, Italy
SO Immunotechnology (1998), 3(4), 233-244
CODEN: IOTEER; ISSN: 1380-2933
PB Elsevier Science B.V.
DT Journal; **General Review**
LA English
AB A review with 123 refs. Recent evidence has been accumulated to suggest that **allergen**-reactive type 2 helper T cells (Th2) play a triggering role in the activation and/or recruitment of IgE antibody-producing B cells, **mast cells** and eosinophils, i.e. the cellular triad involved in the **allergic** inflammation. Interleukin (IL)-4 prodn. by a still unknown cell type (T cell subset, **mast cell**/basophil) at the time of antigen presentation to the Th cell is crit. for the development of Th2 cells. Other **cytokines**, such as IL-1 and IL-10, and hormones, such as calcitriol and progesterone, also play a favoring role. In contrast, **cytokines** such as interferon (IFN-.alpha.), IFN-.gamma., IL-12 and transforming growth factor (TGF)-.beta., and hormones, play a neg. regulatory role on the development of Th2 cells. However, the mechanisms underlying the preferential activation by environmental **allergens** of Th2 cells in atopic individuals still remain obscure. Some gene products selectively expressed in Th2 cells or selectively controlling the expression of IL-4 have recently been described. Moreover, **cytokines** and other gene products that dampen the prodn. of IL-4, as well as the development and/or the function of Th2 cells, have been identified. These findings allow us to suggest that the up-regulation of genes controlling IL-4 expression and/or abnormalities of regulatory mechanisms of Th2 development and/or function may be responsible for Th2 responses against common environmental **allergens** in atopic people. The new insights in the pathophysiol. of T cell responses in atopic diseases provide exciting opportunities for the development of novel immunotherapeutic strategies. They include the induction of nonresponsiveness in **allergen**-specific Th2 cells by **allergen** peptides or redirection of **allergen**-specific Th2 responses by Th1-inducing **cytokines**, altered peptide ligands, **allergens** incorporated into recombinant microorganisms or bound to appropriate adjuvants, and plasmid DNA vaccination. In severe atopic patients, the possibility of nonallergen-specific immunotherapeutic regimens designed to target Th2 cells or Th2-dependent effector mols., such as specific IL-4 transcription factors, IL-4, IL-5 and IgE, may also

be suggested.

L7 ANSWER 23 OF 12[redacted] CAPLUS COPYRIGHT 1999 ACS
AN 1998:218490 CAPLUS
DN 128:320225
TI Mechanisms of the inflammatory **allergic** reaction in the respiratory tract. Recent advances
AU Tonnel, Andre Bernard; Capron, Monique
CS Unites INSERM, Institut Pasteur Lille, Lille, 59019, Fr.
SO Bull. Acad. Natl. Med. (Paris) (1997), 181(8), 1563-1574
CODEN: BANMAC; ISSN: 0001-4079
PB Academie Nationale de Medecine
DT Journal; **General Review**
LA French
AB A review with 35 refs. Mechanisms of the **allergic** inflammatory reaction in the respiratory tract, such as those involved in chronic asthma and **allergic** rhinitis involve a complex and integrated cellular cascade. After **allergen** inhalation, the response is initiated by the airway epithelial dendritic cells which are responsible after transportation to regional lymph nodes, for the presentation to naive CD4+ T helper (Th0) cells. After the first phase of **allergen** sensitization, CD4+ T cells give rise in atopic patients to lymphocytes CD4+ with a **cytokine** profile of Th2-type (secretion of IL-4, IL-5 and IL-13). Both **cytokines** IL-4 and IL-13 favor the IgE antibody prodn. towards inhaled **allergens**, while IL-5 allows the differentiation, activation and survival of eosinophils. Among effector cells, two are predominant: **mast cells** and activated eosinophils. During the initiation phase of the **allergic** reaction the dominant phenomena is represented by IgE mediated **mast cell**/basophil activation which leads to the release of granular **mast cell** mediators but also to the secretion by **mast cells** of **cytokines** also offering a Th2 profile. Moreover in chronic asthma histopathol., immunocytochem. studies and in situ hybridization techniques demonstrate a lage recruitment of inflammatory cells, mainly of activated eosinophils that similarly produce Th2 type **cytokines** and participate in the development of the local **allergic** inflammation. Several additive environmental factors such as viruses or aeropollutants are susceptible to amplify the Th2 type response; conversely specific immunotherapy has been shown to allow a shift from the Th2 to Th1 profile, which explains at least for a part its therapeutic effects.

L7 ANSWER 24 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1998:202807 CAPLUS
DN 128:229269
TI Histamine in the pathogenesis of asthma
AU Akagi, Masaaki
CS Fac. Pharm. Sci., Tokushima Bunri Univ., Tokushima, 770-8514, Japan
SO Nippon Yakurigaku Zasshi (1998), 111(4), 217-222
CODEN: NYKZAU; ISSN: 0015-5691
PB Nippon Yakuri Gakkai
DT Journal; **General Review**
LA Japanese
AB A review with 47 refs. While it is clear that the clin. expression of IgE-mediated diseases depends upon the actions of multiple mediators, histamine, the earliest recognized mediator of **allergy**, remains a prominent contributor. Histamine released from **mast cells** binds to specific receptors (H1, H2, H3) to produce its clin. effects. The cardinal features of asthma include smooth muscle spasm, mucosal edema, inflammation, and mucus secretion. It has been demonstrated that 2 of these features, bronchospasm and mucosal edema, can be caused by H1-receptor stimulation, while H2- and possibly H1-activation

are probably minor causes of mucus secretion. Histamine interacts directly with the endothelial cells (EC) and induces permeability, a transient expression of P-selectin and the secretion of lipid mediators (e.g. PGI2, PAF and LTB4). Moreover, histamine induces a significant increase of IL-6 and IL-8 secretion by EC. Since IL-8 exerts a chemotactic activity for neutrophils, eosinophils, and basophils, and IL-6 is involved in endothelium permeability, the secretion of **cytokines** may be involved in the late phase reaction. Some antihistamines (i.e., levocabastine, terfenadine, loratadine, azelastine, and oxatomide) can reduce ICAM-1 expression. The participation of histamine in the **allergic** inflammation, including asthma, must be re-examd., since the effects of histamine are more widespread.

L7 ANSWER 25 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1998:145067 CAPLUS
DN 128:242652
TI Role of **cytokines** and chemokines in the late phase **allergic** reaction
AU Ebisawa, Motohiro; Tachimoto, Hiroshi; Likura, Yoji; Akiyama, Kazuo; Saito, Hirohisa
CS Clinical Research Center for Allergy, National Sagamihara Hospital, Kanagawa, Japan
SO Prog. Allergy Clin. Immunol., Proc. Int. Congr. Allergol. Clin. Immunol., 16th (1997), 1-6. Editor(s): Oehling, Albert K.; Huerta Lopez, J. G. Publisher: Hogrefe & Huber, Seattle, Wash.
CODEN: 65SQAB
DT Conference; **General Review**
LA English
AB A review with 40 refs. Recent advances in our understanding of late phase **allergic** reactions (LPR) have been made by enhancements in our knowledge of **cytokines**, chemokines, and cell adhesion mols. Although **cytokines** produced by **mast cells** and Th2 cells are thought to be involved in the recruitment of inflammatory cells, particularly eosinophils, the **mast cell**-derived **cytokines** and Th2 cell-derived **cytokines** may contribute to the formation of LPR sequentially. The **mast cell cytokines** may function as triggers for the classical LPR, and Th2 **cytokines** for the sustained events after LPR with more pure eosinophil infiltration. The selective eosinophil recruitment to the sites of **allergic** reactions is coordinated by three classes of **cytokines**. The first class of **cytokines**, IL-1.beta., TNF-.alpha., IL-4 and IL-13, upregulates the vascular cell adhesion mols. The second category of **cytokines** is the eosinophil-active **cytokines** such as IL-5, GM-CSF, and IL-3, which prevent apoptosis and control the activation status of eosinophils at the inflamed sites. The third class, chemokines, attracts eosinophils to the sites of **allergic** inflammation. The source of chemokines varies among many cell types, however, recent studies indicate that one major source is airway epithelial cells.

L7 ANSWER 26 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1998:82573 CAPLUS
DN 128:179059
TI An update on interleukin-4 and its receptor
AU Chomarat, Pascale; Banchereau, Jacques
CS Baylor Institute for Immunology Research, Dallas, TX, 75246, USA
SO Eur. Cytokine Network (1997), 8(4), 333-344
CODEN: ECYNEJ; ISSN: 1148-5493
PB John Libbey Eurotext
DT Journal; **General Review**
LA English
AB A review with 79 refs. Interleukin-4 (IL-4) exhibits many biol. and

immunoregulatory functions on B lymphocytes, monocytes, dendritic cells and fibroblasts. The IL-4 gene is located on chromosome 5 and displays several cell-specific regulatory sequences in its promoter, which explain its restricted secretion pattern to activated T cells and **mast cells**. The IL-4 receptor is multimeric and is constituted by at least IL-4R.alpha., a chain common to other **cytokine** receptors. Two types of IL-4 receptors have been defined: one constituted by the IL-4R.alpha. and the .gamma.c chain, and a second constituted by the IL-4R.alpha. and the IL-13R.alpha., which is able to transduce both IL-4 and IL-13 signals. Major events of IL-4 transducing signal have now been elucidated and are known to be mediated through JAK/IRS-2 and STAT6 pathways. Numerous studies have also demonstrated the key regulatory role of IL-4 in **allergic** responses as well as its anti-tumor and anti-inflammatory effects.

L7 ANSWER 27 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1998:30925 CAPLUS
DN 128:175645
TI Drug therapy for ocular **allergy**
AU Hingorani, Melanie
CS Moorfields Eye Hospital, London, UK
SO Expert Opin. Invest. Drugs (1998), 7(1), 27-55
CODEN: EOIDER; ISSN: 0967-8298
PB Ashley Publications
DT Journal; **General Review**
LA English
AB A review with 248 refs. The spectrum of ocular **allergy** ranges from mild, non-sight threatening disease, such as hay fever, to disorders such as atopic keratoconjunctivitis (AKC) which cause permanent ocular surface changes and reduced vision. The ideal treatment is with topical prepns. Launched topical prepns. include antihistamines and **mast cell** (MC) stabilizers, which are safe, but only moderately potent, steroids, which are very potent, but carry very serious side-effects, and cyclosporin A, which is not widely available and difficult to tolerate. There are a no. of antihistamines, MC stabilizers (and combinations thereof) and steroids in development which are of potential interest. Other possibilities for therapeutic intervention include inhibition of tryptase, cyclooxygenase (COX), leukotrienes (LTs), bradykinins (BKs), platelet activating factor (PAF) and IgE. Therapies based on **cytokine** antagonism and agonism, T-cell inhibition and adhesion mol. antagonism might be expected to provide safe, but potent new modes of treatment. The increasing interest in research into the pathogenesis of ocular **allergic** inflammation may lead to more relevant approaches, such as eosinophil inhibition. Success will be highly dependent on the ability to produce suitable topical ophthalmic prepns.

L7 ANSWER 28 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:811229 CAPLUS
DN 128:73981
TI The cells of the **allergic** response; **mast cells**, basophils, and eosinophils
AU Costa, John J.; Weller, Peter F.; Galli, Stephen J.
CS USA
SO JAMA, J. Am. Med. Assoc. (1997), 278(22), 1815-1822
CODEN: JAMAAP; ISSN: 0098-7484
PB American Medical Association
DT Journal; **General Review**
LA English
AB A review with 69 refs. **Mast cells**, basophils, and eosinophils have long been regarded as important effector cells in **allergic** disorders. Indeed, it is thought that the cells' cytoplasmic granule-assocd. or lipid mediators contribute to many of the signs and symptoms that are characteristic of these diseases.

Mast cells, basophils, and eosinophils also probably contribute to protective host responses, esp. to parasites. In addn., recent evidence shows that **mast cells**, basophils, and eosinophils can secrete a wide spectrum of **cytokines** and, in some cases, express functions that may permit them to regulate the development or perpetuation of **allergic** responses. Thus, **mast cells**, basophils, and eosinophils may express immunoregulatory activities, as well as serve as effector cells.

L7 ANSWER 29 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:772782 CAPLUS
DN 128:47004
TI The **mast cell** and molecular mechanisms of **allergy**
AU Saito, Hirohisa
CS Shoni Iryo Kenkyu Senta, Kokuritsu Shoni Byoin, Tokyo, 154, Japan
SO Mol. Med. (Tokyo) (1997), 34(12), 1478-1487
CODEN: MOLMEL; ISSN: 0918-6557
PB Nakayama Shoten
DT Journal; **General Review**
LA Japanese
AB A review with 33 refs. Differentiation pathways of **mast cells** (MC) in mouse and human, and participating mols. in differentiation and activation of MC are discussed. Interleukin 3 (IL-3) supports the differentiation of MC from hematopoietic cells in mouse, and human hematopoietic cells require steel factor (c-kit, stem cell factor) in the differentiation to MC in addn. to IL-3. Human MC are derived from CD34+CD38+ cells. MC secrete various **cytokines** after several hours of **allergen** challenge, which induces or helps selective penetration of eosinophils. Intracellular signal transduction pathway via Fc.epsilon. receptor I (Fc.epsilon.RI) in MC is depicted. IgE sensitization alone enhances MC function after 48 h, and IL-4 enhances the effects of IgE. IL-4 increases expression of Fc.epsilon.RI and histamine release. MC may play central roles in exacerbation process of **allergic** reaction by prodn. of large amts. of Th2 **cytokines**.

L7 ANSWER 30 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:748225 CAPLUS
DN 128:33425
TI Molecular and cellular biology of **mast cells** and basophils
AU Marone, Gianni; Casolaro, Vincenzo; Patella, Vincenzo; Florio, Giovanni; Triggiani, Massimo
CS Division of Clinical Immunology and Allergy, University of Naples Federico II, Naples, Italy
SO Int. Arch. Allergy Immunol. (1997), 114(3), 207-217
CODEN: IAAIEG; ISSN: 1018-2438
PB Karger
DT Journal; **General Review**
LA English
AB A review with 118 refs. In all mammalian species investigated so far, **mast cells** and basophils are the only cells that synthesize histamine and express plasma membrane receptors that bind IgE with high affinity (Fc.epsilon.RI). Human basophils and **mast cells** derive from distinct precursors that originate in the bone marrow and fetal liver and probably circulate in peripheral blood. There is extensive evidence that **mast cells** and basophils and their mediators are primary effectors of **allergic** inflammation. Immunol. activated human basophils release two **cytokines**: IL-4 and IL-13. Expression of several **cytokines** has been documented in a no. of exptl. models of human and rodent **mast cells**. However, to date few studies

have analyzed the mechanisms of gene expression in human Fc.epsilon.RI+ cells. Some of these studies imply a role for NFAT and GATA family members in the I[redacted]-mediated activation of **cytokin**[redacted]gene transcription in basophils and **mast cells**. Studies of human basophils and **mast cells** isolated from different anat. sites have established the different profiles of eicosanoids released by these cells. Recently, the characterization of arachidonic acid pools and the identification of novel enzymes involved in arachidonate remodeling and mobilization clarified in part how eicosanoid productions is regulated in **mast cells** and basophils. In addn. to histamine, human **mast cell** secretory granules contain the neutral proteases tryptase, chymase and carboxypeptidase that possess several biochem. properties. In particular, tryptase may play a role as a fibrogenic factor and chymase might convert angiotensin I to angiotensin II. **Mast cells** are present in human heart and in human coronary arteries raising the possibility that local activation of cardiac **mast cells** might contribute to certain cardiovascular diseases. Recent evidence also suggests that **mast cells** and basophils can play a role during viral and bacterial infections. It is now evident that in man these two cells not only participate in inflammation assocd. with **allergic** disease, but also in chronic and fibrotic disorders affecting several organs and in host defense against bacterial and viral infections.

L7 ANSWER 31 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:733128 CAPLUS
DN 128:12403
TI Immunologic basis of chronic **allergic** diseases: clinical messages from the laboratory bench
AU Leung, Donald Y. M.
CS Div. Pediatric Allergy and Immunology, Natl. Jewish Med. and Res. Center, Denver, CO, 80206, USA
SO Pediatr. Res. (1997), 42(5), 559-568
CODEN: PEREBL; ISSN: 0031-3998
PB Williams & Wilkins
DT Journal; **General Review**
LA English
AB A review with 115 refs. During the past decade there have been many advances in the understanding of the mechanisms underlying **allergic** response. Immediate hypersensitivity reactions are mediated primarily by **mast cells** in an IgE-dependent manner. After the local release of various mediators, proinflammatory **cytokines**, and chemokines, there is a cell-mediated response that is dominated by eosinophils and T lymphocytes. The majority of T cells in early **allergic** reactions are memory T cells secreting helper type 2 (TH2)-like **cytokines**, i.e. IL-4, IL-5, and IL-13, but not interferon-.gamma.. These **cytokines** regulate IgE synthesis and promote eosinophil differentiation and cell survival, thus contributing to **allergic** inflammatory responses. Failure to control immune activation early in the course of **allergic** inflammation may blunt the response to glucocorticoid therapy and contribute to longterm morbidity of disease. The identification of key cells and **cytokines** involved in the initiation and maintenance of **allergic** inflammation is likely to become an important therapeutic target in the future management of this important group of diseases.

L7 ANSWER 32 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:725933 CAPLUS
DN 128:46903
TI Etiological mechanism of **allergic** rhinitis
AU Otsuka, Hirokuni
CS Second Hospital, Nippon Med. Sch., Japan

SO Arerugi (1996), 152-153. Editor(s): Ra, Chisei. Publisher: Yodosha, Tokyo, Japan.
CODEN: 65GGAN
DT Conference; **General Review**
LA Japanese
AB A review with 16 refs., on structure of nose, pathol. mechanism, distribution and function of **mast cell** in nasal mucosa, and nose mucosal **cytokine** and cell infiltration.

L7 ANSWER 33 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:723017 CAPLUS
DN 128:46440
TI Dermatological **allergies** and **cytokines**
AU Morita, Eishin; Yamamoto, Shoso
CS Fac. Med., Hiroshima Univ., Japan
SO Arerugi to Saitokain (1996), 219-226. Editor(s): Miyamoto, Terumasa; Ishikawa, Takeru; Iikura, Yoji. Publisher: Iyaku Janarusha, Osaka, Japan.
CODEN: 65GJAW
DT Conference; **General Review**
LA Japanese
AB A review with 15 refs., on **cytokines** relate to IgE prodn. and **cytokines** relate to proliferation and differentiation of **mast cells**, skin Langerhans' cell and IgE, and eosinophil and skin **allergy**.

L7 ANSWER 34 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:700406 CAPLUS
DN 127:345010
TI **Mast cells** as potent inflammatory cells
AU Dumitrascu, Diana
CS Third Medical Dep., Univ. Medicine & Pharmacy, Cluj-Napoca, 3400, Rom.
SO Rev. Roum. Med. Interne (1996), 34(3-4), 159-172
CODEN: RRINEH; ISSN: 1220-4749
PB Editura Academiei Romane
DT Journal; **General Review**
LA English
AB A review with 30 refs. In the last decade, new information was achieved on **mast cells** (MC). Their origin is assumed to be different from that of the basophils. There are two types of MC with differences in structure, distribution and function: conjunctival and mucosal. MCs are among the most important cells in the development of **allergic** inflammation through the **cytokines** and mediators released on the activation of the surface receptors (high-affinity receptors for IgE:Fc.epsilon.RI). The **cytokines** released by MCs, e.g., interleukin 5 (IL5), IL8, are chemoattractants for eosinophils and neutrophils, resp. The two types of mediators released by MC are those preformed, such as histamine, tryptase, serotonin, and the newly-synthesized ones, such as prostaglandin D2 (PGD2), leukotrienes C4 (LTC4), D4 (LTD4), E4 (LTE4), induce vasodilatation, bronchoconstriction, cellular chemotaxis, increase vascular permeability. The involvement of MC in many human diseases was shown within in vivo and in vitro studies (in **allergy**, lung fibrosis, atherosclerosis, carcinogenesis, etc.).

L7 ANSWER 35 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:654318 CAPLUS
DN 127:329917
TI **Cytokines** as targets for the inhibition of eosinophilic inflammation
AU Hogan, Simon P.; Foster, Paul S.
CS CELLULAR SIGNAL TRANSDUCTION LABORATORY, THE DIVISION OF BIOCHEMISTRY AND MOLECULAR BIOLOGY, THE JOHN CURTIN SCHOOL OF MEDICAL RESEARCH, AUSTRALIAN NATIONAL UNIVERSITY, CANBERRA, 0200, Australia

SO Pharmacol. Ther. (1997), 74(3), 259-283
CODEN: PHTHDT; ISSN: 0163-7258
PB Elsevier
DT Journal; **General Review**
LA English
AB A review with 192 refs. Eosinophilic inflammation is thought to play a central role in the pathogenesis of asthma. The immunoregulatory effects of interleukin (IL)-4, IL-5 and IgE suggest that these mols. play key roles in the effector function of eosinophils and **mast cells**. IL-4 regulates the development of CD4+ TH2-type cells, which elicit essential signals through IL-4 and IL-5 for the regulation of IgE prodn. and eosinophilia, resp. IL-5-regulated pulmonary eosinophilia and airways dysfunction can also occur independently of IL-4 and **allergen**-specific Igs. Such IL-4-independent pathways may also play a substantive role in the etiol. of asthma. Thus, evidence is now emerging that **allergic** airways disease is regulated by humoral and cell-mediated components. The essential and specific role of IL-5 in regulating eosinophilia, and the subsequent involvement of this leukocyte in the induction of lung damage and airways dysfunction, identifies IL-5 as a primary therapeutic target for the relief of airways dysfunction in asthma.

L7 ANSWER 36 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:640877 CAPLUS
DN 127:291746
TI Bronchial mucosal **mast cell**
AU Takahashi, Kiyoshi
CS Kokuritsu Ryoyosho Minami Okayama Byoin, Okayama, 701-03, Japan
SO Arerugi no Ryoiki (1997), 4(10), 1382-1387
CODEN: ARRYFB; ISSN: 1340-2358
PB Iyaku Janarusha
DT Journal; **General Review**
LA Japanese
AB A review with 13 refs. on distribution of **mast cells** in asthma, on correlation of **mast cells** with basophils in asthma, on roles of bronchial mucosal **mast cells** in airway inflammatory reactions, and on a new role of **mast cells** and basophils in **allergic** inflammatory reactions.

L7 ANSWER 37 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:640876 CAPLUS
DN 127:329960
TI Nasal mucosal **mast cell**
AU Kawabori, Shinichi
CS Jibi Inkoka, Asahikawa Ika Daigaku, Asahikawa, 078, Japan
SO Arerugi no Ryoiki (1997), 4(10), 1375-1381
CODEN: ARRYFB; ISSN: 1340-2358
PB Iyaku Janarusha
DT Journal; **General Review**
LA Japanese
AB A review with 19 refs. on **cytokines** produced by nasal mucosal **mast cells** in **allergic** rhinitis, on mechanisms of nasal mucosal **mast cell** proliferation in **allergic** rhinitis, on involvement of nasal mucosal **mast cells** in IgE prodn. in **allergic** rhinitis, and on IgE receptor expressed by nasal **mast cells** in **allergic** rhinitis.

L7 ANSWER 38 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:640874 CAPLUS
DN 127:291745
TI Delayed **allergic** reactions and **mast cell**
AU Ebisawa, Motohiro; Tachimoto, Hiroshi; Saito, Hirohisa

CS Rinsho Kenkyubu, Kokuritsu Sagamihara Byoin, Sagamihara, 228, Japan
SO Arerugi no Ryoiki (1997), 4(10), 1359-1365
CODEN: ARRYFB; [redacted]N: 1340-2358
PB Iyaku Janarusha
DT Journal; **General Review**
LA Japanese
AB A review with 18 refs. on past and recent findings about the roles of human or mouse **mast cells** in delayed **allergic** reactions.

L7 ANSWER 39 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:640870 CAPLUS
DN 127:291743
TI **Allergy** and **mast cell**, basophil
AU Morita, Yutaka
CS Igakubu, Tokyo Daigaku, Tokyo, 113, Japan
SO Arerugi no Ryoiki (1997), 4(10), 1329-1333
CODEN: ARRYFB; ISSN: 1340-2358
PB Iyaku Janarusha
DT Journal; **General Review**
LA Japanese
AB A review with 6 refs. on the roles of **mast cells** and basophils in **allergic** reaction. Specifically, the roles of basophils and **cytokines** produced from the **mast cells** in the immediate asthmatic response (IAR) and the late asthmatic response (LAR) were discussed.

L7 ANSWER 40 OF 122 CAPLUS COPYRIGHT 1999 ACS
AN 1997:611491 CAPLUS
DN 127:219097
TI Progress in study of pathological mechanisms and treatment of bronchial asthma
AU Ito, Koji
CS Naika Butsuri Ryohogaku, Tokyo Daigaku, Tokyo, 113, Japan
SO Nippon Naika Gakkai Zasshi (1997), 86(9), 1748-1756
CODEN: NNGAAS; ISSN: 0021-5384
PB Nippon Naika Gakkai
DT Journal; **General Review**
LA Japanese
AB A review with 16 refs., discussing: (1) immediate and late asthmatic responses, (2) roles of **mast cell**, eosinophil, T cell, and bronchial epithelial cell in bronchial contraction and airway inflammation in atopic bronchial asthma, (3) pathogenesis of nonatopic asthma and aspirin-induced asthma, (4) roles of **cytokines** and adhesion mols. in pathogenesis of bronchial asthma, and (5) treatment of chronic asthma with bronchodilators, antiallergic agents, inhaled steroids, and immunosuppressants.

AN 1997:700406 CAPLUS
DN 127:345010
TI **Mast cells** as potent inflammatory cells
AU Dumitrascu, Diana
CS Third Medical Dep., Univ. Medicine & Pharmacy, Cluj-Napoca, 3400, Rom.
SO Rev. Roum. Med. Interne (1996), 34(3-4), 159-172
CODEN: RRINEH; ISSN: 1220-4749
PB Editura Academiei Romane
DT Journal; **General Review**
LA English
AB A review with 30 refs. In the last decade, new information was achieved on **mast cells** (MC). Their origin is assumed to be different from that of the basophils. There are two types of MC with differences in structure, distribution and function: conjunctival and mucosal. MCs are among the most important cells in the development of **allergic** inflammation through the **cytokines** and mediators released on the activation of the surface receptors (high-affinity receptors for IgE:Fc.epsilon.RI). The **cytokines** released by MCs, e.g., interleukin 5 (IL5), IL8, are chemoattractants for eosinophils and neutrophils, resp. The two types of mediators released by MC are those preformed, such as histamine, tryptase, serotonin, and the newly-synthesized ones, such as prostaglandin D2 (PGD2), leukotrienes C4 (LTC4), D4 (LTD4), E4 (LTE4), induce vasodilatation, bronchoconstriction, cellular chemotaxis, increase vascular permeability. The involvement of MC in many human diseases was shown within in vivo and in vitro studies (in **allergy**, lung fibrosis, atherosclerosis, carcinogenesis, etc.).

AN 1997:700406 CAPLUS
DN 127:345010
TI **Mast cells** as potent inflammatory cells
AU Dumitrascu, Diana
CS Third Medical Dep., Univ. Medicine & Pharmacy, Cluj-Napoca, 3400, Rom.
SO Rev. Roum. Med. Interne (1996), 34(3-4), 159-172
CODEN: RRINEH; ISSN: 1220-4749
PB Editura Academiei Romane
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